The Use of Transcutaneous Oxygen Tension Measurements in the Diagnosis of Peripheral Vascular Insufficiency

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Transcutaneous tissue oxygen tension ($PtcO_2$) was evaluated as a noninvasive diagnostic test for peripheral arterial insufficiency; $PtcO_2$ was measured at rest, during exercise, and following exercise at three leg sites in 36 controls and 138 patients with exercise-induced leg pain. Resting foot $PtcO_2$ differed significantly in controls and in patients with rest pain (32/138) and was 60.1 ± 6.82 mmHg and 3.66 ± 3.68 mmHg, respectively ($p < 0.001$). Abnormal resting $PtcO_2$ values occurred in 80% of claudicants (85/138) and 20% had normal values. However, all vascular claudicants exhibited a decline in $PtcO_2$ following exercise, a finding that distinguished them from controls ($p< 0.001$). The $PtcO_2$ values in 21 patients who were subsequently shown not to have vascular disease did not differ significantly from controls ($p > 0.5$). Comparison with angiograms (48) showed that $PtcO_2$ following exercise had a 100% sensitivity and specificity in detecting the presence of arterial disease. If resting values alone are considered, sensitivity falls to 77%. This study demonstrates that measurement of $PtcO_2$ at rest and particularly after exercise is a simple and sensitive noninvasive diagnostic test for peripheral arterial insufficiency. This test will serve to distinguish between vascular and other causes of exercise-induced leg pain.

THE ABILITY TO MEASURE the partial pressure of tissue oxygen by transcutaneous means has placed a new, noninvasive, investigational tool at the disposal of the vascular surgeon. Using this technique it should be possible not only to diagnose the presence of peripheral vascular disease but also to quantitate objectively its severity. In addition, the technique should provide the ability to monitor noninvasively the progress of the disease and to assess the results of surgical intervention.

In 1972 Huch, Huch, and Menzer[1] and Eberhard, Mindt, and Hammacher[2] simultaneously published abstracts on the use of a miniaturized Clark electrode.

Although the technique has been available for more than a decade transcutaneous tissue oxygen tension ($PtcO_2$) has not made a large impact in the field of vascular surgery. Papers by Matsen et al.,[3] Franzeck et al.,[4] and White et al.,[5] represent the initial impact of this technique on vascular surgery. These papers essentially examine resting $PtcO_2$ in patients with peripheral vascular disease and in controls and show that $PtcO_2$ is reduced in the presence of arterial disease. Recently Hauser and Shoemaker[6] studied $PtcO_2$ in 12 patients with peripheral vascular disease and in 12 controls and found $PtcO_2$ to be a sensitive and specific test for arterial insufficiency. Burgess et al.[7] used $PtcO_2$ to predict the outcome in patients requiring amputation.

Oxygen delivery is a major function of the circulation and $PtcO_2$ measurement in the lower limb might therefore provide a noninvasive means by which the peripheral circulation can be assessed. Measurement of $PtcO_2$ can be made continuously both at rest and during exercise and may thus give a more accurate assessment of the state of the peripheral circulation. We therefore undertook a study in which we examined the application of $PtcO_2$ measurements at rest, during exercise, and following exercise in the diagnosis of peripheral arterial insufficiency. The results of these studies in 174 individuals are reported.

Method

Transcutaneous tissue oxygen measurements were made using a Clark-type electrode with a silver ring anode and a large gold cathode (diameter—2.4 mm) containing a heating resistor and two calibrated precision thermistors. The sensor is prepared by applying a special electrolyte solution and a precut membrane. The cathode is polarized with −600 mv, and PO_2 measurement is based on the electrochemical reduction of oxygen. The sensor is calibrated using a one-point calibration with air. A two-point calibration using nitrogen gas for zero calibration can be utilized if necessary. Daily calibration of the sensor was carried out and repeated prior to undertaking any study. The sensor was attached to a Kontron 632 module that produced a digital readout of the $PtcO_2$ and was, in turn, connected to a Kontron 3300 recorder for contin-

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TABLE 1. *Ranges and Means of $PtcO_2$ in Control Subjects (N = 36)*

	Supine	Erect	Exercise	1 Min. Postexercise	5 Min. Postexercise	10 Min. Postexercise
Above knee	65.8 ± 7.83* (52–79)	74.5 ± 6.56 (63–87)	78.1 ± 6.09 (65–94)	74.9 ± 6.47 (60–86)	73.2 ± 6.03 (63–85)	70.4 ± 7.62 (58–85)
Below knee	63.3 ± 7.83 (50–84)	73.2 ± 7.73 (58–88)	77.6 ± 8.05 (62–92)	71.4 ± 7.51 (56–85)	69.8 ± 7.21 (50–84)	66.2 ± 8.75 (51–84)
Foot	60.1 ± 6.82 (50–77)	70.7 ± 7.39 (58–85)	75.3 ± 8.60 (59–90)	69.0 ± 7.49 (53–84)	68.1 ± 6.86 (58–82)	64.6 ± 7.50 (53–83)

Values expressed in mmHg.

* Standard deviation.

uous tracing of the results obtained. The equipment is commercially available from Kontron, Mississauga, Ontario.

The electrodes were applied to the skin by way of double-sided adhesive rings and were placed on the anterior aspect of the leg 10 cm above and 10 cm below the knee and on the dorsum of the foot. A fourth electrode was, in some instances, positioned on the chest 5 cm below the mid-point of the left clavicle for control readings. Following application of the electrodes, the patient remained supine for 10 to 15 minutes for equilibration. The individual then stood and the electrodes were allowed to equilibrate again over 3 to 5 minutes, following which the patient exercised on a treadmill inclined to 20 degrees at a speed of 1.5 miles per hour, for a maximum of 5 minutes or until leg pain prevented further exercise. On cessation of exercise a supine position was resumed for a minimum of 10 minutes. Readings were recorded from the initial supine position, following 3 to 5 minutes of standing, on completion of exercise and at 1-, 5-, and 10-minute intervals following exercise. In addition, a continuous tracing of the $PtcO_2$ measurements during the procedure was made. All $PtcO_2$ measurements were made with the sensor temperature set at 45 C with the patient breathing room air.

The investigation was carried out on a control group and a study group. The control group included both men and women who had no clinical history or symptoms of vascular disease and who had normal pulses present in both lower limbs. The study group consisted of patients referred for investigation of exercise-induced leg pain. One leg was studied at a time and the symptomatically worst leg was chosen in each instance. Data were analyzed using an unpaired t-test.

Results

Thirty-six control subjects and 138 patients with exercise-induced leg pain were studied. The control group included 17 men and 19 women from 25 to 79 years old with a mean age of 49.6 ± 16.97 years. Thirteen controls were less than 40 years of age (mean 29.8 ± 3.81 years) and 23 were older (mean 60.7 ± 9.40 years). Control values are listed in Table 1, which shows the ranges and means of $PtcO_2$ during exercise and rest. There is a gradient of 5.7 mmHg between above-knee and foot $PtcO_2$ values at rest. This gradient decreased with standing and exercise but increased again with the resumption of the supine position following exercise. Mean $PtcO_2$ increased significantly ($p < 0.001$) on standing, the average increase being in the order of 10 mmHg. A further increase of about 4 mmHg occurred during exercise. After the cessation of exercise, $PtcO_2$ decreased slowly towards supine resting values and readings obtained 10 minutes following completion of exercise did not differ significantly ($p > 0.1$) from resting values.

Figure 1 shows a typical $PtcO_2$ tracing from a control subject. Figure 2 shows the mean $PtcO_2$ observed at the three leg sites during rest and exercise.

One hundred and thirty-eight patients with ischemic rest pain or exercise-induced leg pain were studied. On the basis of $PtcO_2$ these patients could be divided into three groups.

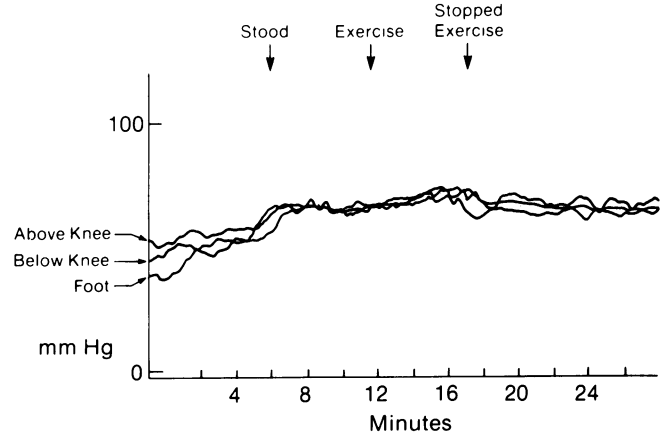

FIG. 1. Recording from a control subject. Note the sharp increase in $PtcO_2$ on standing (first arrow) followed by a more gradual increase during exercise (second arrow) and a slow decline following cessation of exercise (third arrow).

Group 1—Ischemic Pain at Rest

All 32 patients with rest pain had abnormal $PtcO_2$ values at rest at one or more levels with the most marked abnormalities occurring in the foot (Table 2). Foot $PtcO_2$ values were less than 10 mmHg (mean 3.68 ± 3.68 mmHg) in all but one patient and differed significantly from controls ($p < 0.001$). Below-knee $PtcO_2$ was abnormal in 27 of 32 patients (mean 29.09 ± 19.66 mmHg) and differed significantly from controls ($p < 0.001$). Above-knee $PtcO_2$ was abnormal in 14 patients (mean 49.88 ± 13.68). On standing, above-knee $PtcO_2$ increased by an average of 14 mmHg while below knee and foot $PtcO_2$ increased by an average of 20 mmHg. Only 13 patients were able to do any exercise. Figure 3 is a tracing from a patient with rest pain, and a comparison of the mean $PtcO_2$ in controls in patients with rest pain is shown in Figure 4.

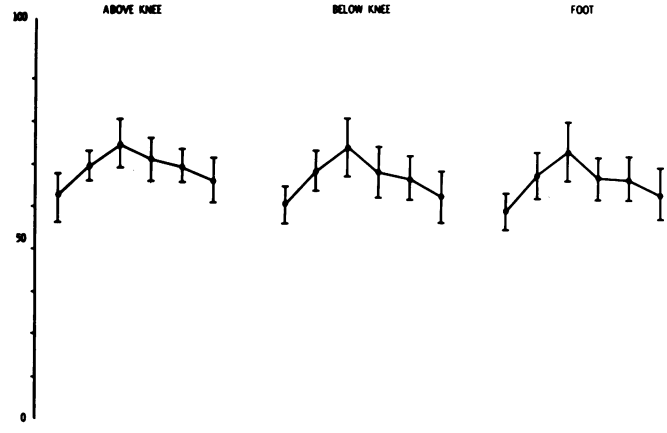

FIG. 2. Mean $PtcO_2$ values at above-knee, below-knee, and foot levels in 36 control subjects at rest, standing, during exercise, and 1, 5, and 10 minutes following exercise.

Group 2—Vascular Claudication

Patients in this group had claudication due to vascular insufficiency and could be divided into two groups on the basis of resting $PtcO_2$ (Table 3). Group 2A included 68 patients who had an abnormal $PtcO_2$ at one or other level in the leg and Group 2B, which included 17 patients in whom resting $PtcO_2$ at all three leg levels lay within the control range. Sixty-five patients were able to exercise and showed a decrease of 5 mmHg above and below the knee and 9 mmHg on the foot. The most striking difference between these patients and controls, however, occurred in the postexercise phase when mean $PtcO_2$ decreased significantly ($p < 0.001$).

Patients in Group 2B had $PtcO_2$ values that did not differ significantly from controls at rest ($p > 0.1$) or when standing ($p > 0.1$), but during and following exercise, below-knee and foot $PtcO_2$ decreased significantly ($p < 0.001$).

Figure 5 and 6 show tracings from patients with claudication. Figure 7 is a comparison of the mean $PtcO_2$ in controls and in claudicants with abnormal $PtcO_2$ at rest. The comparison between controls and claudicants with normal $PtcO_2$ at rest is shown in Figure 8. Both figures demonstrate the abnormal decline in $PtcO_2$ following exercise, which is characteristic of vascular claudication.

Group 3—Non-vasculogenic Claudication

This group included 21 patients who complained of leg pain on exercise; $PtcO_2$ in this group was not significantly different from controls ($p > 0.5$). Figure 9 shows the similarity between the mean $PtcO_2$ found in this group and that observed in controls, and the $PtcO_2$ values are shown in Table 4. The majority of the patients in this group did not have peripheral vascular disease on the basis of normal doppler pressure measurements both at rest and following exercise. Four patients with questionable doppler pressures had arteriograms, all of which were normal. The remaining patients were diagnosed by other means as having neurogenic claudication or arthritic leg pain.

TABLE 2. *Ranges and Means of $PtcO_2$ in Patients with Rest Pain*

	Supine (N = 32)	Erect (N = 30)	Exercise (N = 13)	1 Min. postexercise (N = 13)	5 Min. Postexercise (N = 13)	10 Min. Postexercise (N = 13)
Above knee	49.9 ± 13.68* (16–74)	63.9 ± 11.03 (30–84)	64.8 ± 13.52 (51–84)	58.1 ± 15.56 (33–85)	57.9 ± 14.55 (31–83)	57.2 ± 10.06 (42–78)
Below knee	29.1 ± 19.66 (2–61)	49.8 ± 19.58 (2–76)	50.1 ± 18.55 (23–81)	32.0 ± 21.33 (3–63)	36.5 ± 23.99 (3–67)	39.9 ± 23.82 (2–71)
Foot	3.68 ± 3.68 (0–17)	25.3 ± 20.14 (1–62)	26.3 ± 25.97 (2–80)	5.1 ± 6.54 (1–24)	5.7 ± 9.79 (1–36)	6.0 ± 9.40 (1–36)

Values expressed in mmHg.

* Standard Deviation.

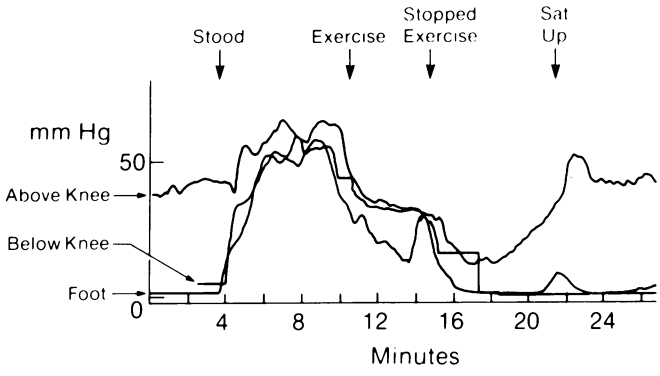

FIG. 3. Recording from a patient with ischemic rest pain showing decreased $PtcO_2$ values at rest that increase on standing (first arrow). Note the decline at all levels during and after exercise (second and third arrows).

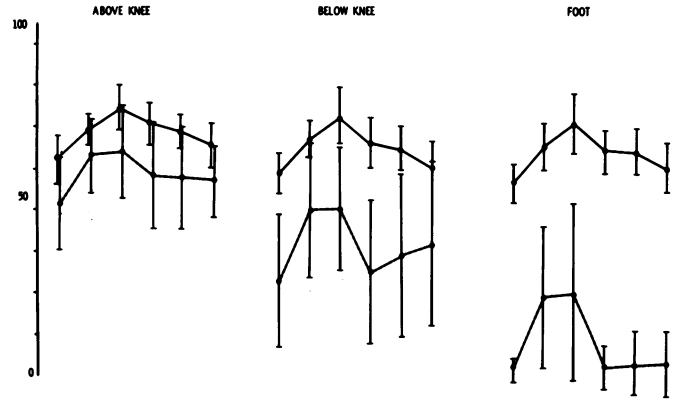

FIG. 4. Comparison of mean $PtcO_2$ in controls (solid line) and in patients with rest pain (stippled line); $PtcO_2$ values at all points differ significantly in both groups ($p < 0.001$, unpaired t-test).

Correlation with Arteriography

Arteriograms were performed in 48 patients, 44 of whom had abnormal $PtcO_2$ studies and four of whom had normal $PtcO_2$ values. The arteriograms were reviewed by one of us (JLP), who was blind to the $PtcO_2$ results and thought that four arteriograms were normal and that the remaining 44 patients had significant arterial disease. The four normal arteriograms belonged to the four patients with normal $PtcO_2$ values. These results give $PtcO_2$ a sensitivity of 100% and a specificity of 100% in the diagnosis of arterial disease. If resting values alone had been considered there would have been 10 false-negatives and no false-positives, giving a sensitivity of 77% and specificity of 100%.

Discussion

The principle of transcutaneous oxygen measurement involves the application of heat to the skin, producing localized hyperaemia and oxygen excess. Oxygen diffuses along a concentration gradient from the capillaries to the tissues and, if still present in excess of skin requirements, may diffuse across the skin where it is electrochemically reduced and measured by a Clark electrode. The amount of oxygen available for diffusion across the skin depends on oxygen delivery, which is a function of arterial oxygen content and blood flow. When blood flow is adequate, $PtcO_2$ follows arterial oxygen content and, conversely, when arterial oxygen content is adequate and flow becomes compromised, $PtcO_2$ follows blood flow.[7,8] While

TABLE 3. *Ranges and Means of $PtcO_2$ in 85 Patients with Vasculogenic Claudication*

	Supine	Erect	Exercise	1 Min. Postexercise	5 Min. Postexercise	10 Min. Postexercise
Group 2A (N = 68)						
Above knee	53.9 ± 7.03* (37–72)	66.1 ± 7.53 (40–81)	61.1 ± 11.30 (31–83)	54.2 ± 16.17 (6–79)	58.4 ± 11.63 (16–77)	59.1 ± 8.10 (41–78)
Below knee	48.2 ± 9.90 (19–67)	61.8 ± 8.66 (34–82)	56.0 ± 11.73 (27–78)	37.1 ± 19.87 (3–73)	47.7 ± 17.39 (0–75)	49.9 ± 14.53 (2–79)
Foot	36.8 ± 12.06 (8–52)	58.0 ± 12.12 (2–77)	49.0 ± 17.60 (10–88)	23.3 ± 19.80 (1–66)	33.3 ± 19.86 (1–67)	40.4 ± 16.95 (1–67)
Group 2B (N = 17)						
Above knee	65.5 ± 7.12 (54–79)	74.3 ± 6.31 (64–86)	71.9 ± 5.63 (64–78)	70.7 ± 8.23 (55–83)	71.6 ± 8.13 (56–87)	70.6 ± 8.46 (57–84)
Below knee	59.1 ± 4.88 (53–69)	69.8 ± 6.58 (60–70)	63.8 ± 9.79 (41–77)	49.1 ± 19.63 (12–70)	54.1 ± 14.26 (34–74)	60.9 ± 5.02 (53–67)
Foot	55.7 ± 4.29 (50–64)	68.2 ± 8.12 (55–82)	53.1 ± 10.08 (29–70)	33.3 ± 15.80 (6–57)	44.7 ± 18.57 (6–66)	52.9 ± 8.74 (37–61)

Values expressed in mmHg.

* Standard Deviation.

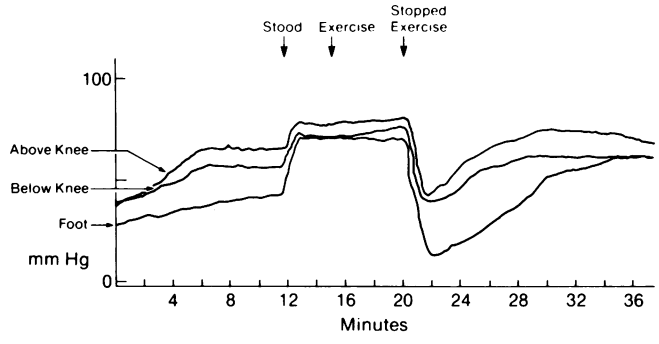

FIG. 5. Recording from a patient with claudication showing decreased foot $PtcO_2$ at rest. There is a sharp decrease in $PtcO_2$ at all leg levels following exercise.

this has been shown to be true in shock it has not been adequately studied in peripheral vascular insufficiency.

In this situation the question to be answered is whether $PtcO_2$ reflects the impairment of blood flow produced by the underlying arterial insufficiency. Both systemic and local factors affect $PtcO_2$ (Table 5). Local factors include skin thickness, capillary density, inflammation, edema, and oxygen consumption of the skin. These may be responsible for the small difference in $PtcO_2$ observed between different sites. They provide a static rather than a dynamic barrier to diffusion of oxygen through the skin and their effects can be minimized by careful selection of electrode sites. Once equilibration has occurred, local factors should not produce further changes in $PtcO_2$.

Systemic factors can be divided into two main groups, those that affect arterial oxygen content and those that affect blood flow. Arterial oxygen content is affected by ventilation and haemoglobin properties. Changes in these variables should produce similar changes in $PtcO_2$ at all three leg sites. Furthermore, none of these factors with the exception of ventilation, which increases during exercise, is likely to change during the course of an individual

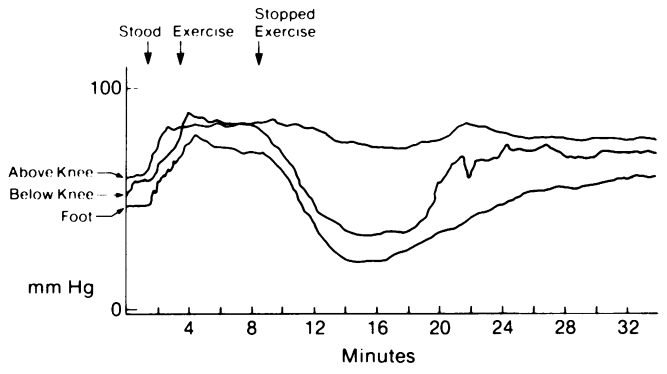

FIG. 6. Recording from a patient with claudication with normal resting $PtcO_2$ values. Below-knee and foot $PtcO_2$ values decline following exercise (third arrow) but the above-knee value does not indicate that $PtcO_2$ may be of value in localizing the level of arterial obstruction.

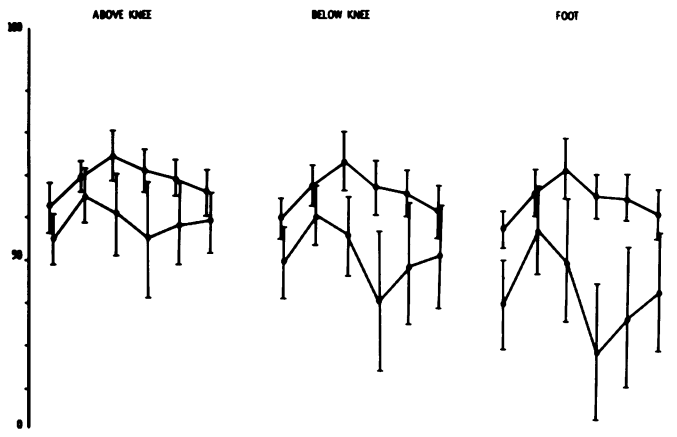

FIG. 7. Comparison of mean $PtcO_2$ values from controls (solid line) and from claudicants with reduced resting $PtcO_2$ values (stippled line). Claudicant $PtcO_2$ values at all points differ significantly from control $PtcO_2$ values ($p < 0.001$, unpaired t-test).

test. The other systemic factor, blood flow, depends on cardiac output and perfusion distribution. Cardiac output, like ventilation, increases during exercise and these two factors are responsible for the increase in $PtcO_2$ that accompanies exercise. Both factors will produce similar changes in $PtcO_2$ from electrodes at any site. The final factor that affects blood flow is perfusion, changes in which may cause variations in readings from $PtcO_2$ electrodes placed where perfusion is likely to be altered. Differences in $PtcO_2$ readings that arise once equilibration has occurred in normally functioning electrodes must therefore be due to changes in perfusion. Depending on whether

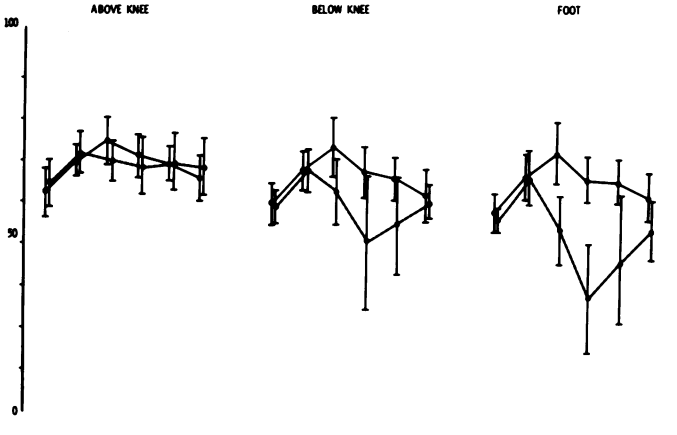

FIG. 8. Comparison of mean $PtcO_2$ from controls (solid line) and from patients with claudication with normal $PtcO_2$ at rest (stippled line); $PtcO_2$ values at rest and on standing do not differ significantly ($p > 0.1$), however, a significant difference occurs during exercise, (above knee–$p < 0.01$, below knee and foot–$p < 0.001$). Following exercise, above-knee values recover rapidly and are not significantly different from controls 1 minute after exercise ($p > 0.3$). Below-knee values recover within 10 minutes ($p > 0.3$), but foot values are still significantly different from controls 10 minutes following exercise ($p < 0.0025$, unpaired t-test).

FIG. 9. Comparison of mean $PtcO_2$ from controls (solid line) and from patients shown not to have peripheral arterial disease (stippled line); $PtcO_2$ values in these two groups of patients do not differ significantly at any point ($p > 0.3$).

the arteriosclerotic lesion is in the iliac, femoral, or tibial vessels, changes in blood flow will result in alterations of $PtcO_2$ readings by one or all of the leg sensors. Therefore, $PtcO_2$ is a variable that reflects the underlying condition.

The question of a control chest electrode must be discussed. When the study was started a control electrode was an integral part of the procedure. A control electrode is necessary to ensure that systemic factors are not responsible for low $PtcO_2$ readings in the leg. Hauser and Shoemaker[6] expressed $PtcO_2$ as a percentage of chest $PtcO_2$ and called the resulting figure the "Regional Perfusion Index." It became apparent, however, that it was not required in most cases because the above-knee value in 85% of patients lay inside the control range at rest. When the above-knee value is low at rest, it then becomes important to measure $PtcO_2$ at an alternate control site to ensure that low $PtcO_2$ readings are not present because of cardiorespiratory insufficiency or anaemia.

There are many causes of exercise-induced leg pain[9] and at times it may be difficult to differentiate them. It appears that $PtcO_2$ is a method by which vascular insufficiency can be distinguished noninvasively from other causes. The increases in Groups 1 and 2 reflect not only the increase in perfusion pressure but also a degree of hyperemia following the relative hypoxia when supine. In controls, $PtcO_2$ increases during exercise and decreases slowly following exercise, reflecting the changes that occur in cardiac output. In patients with vascular disease the increased cardiac output during exercise together with the increased perfusion pressure in the erect posture almost overcome the effect of the arterial insufficiency. However, when these patients stop exercising and lie down the reductions in cardiac output and perfusion pressure result in decreased blood flow in the leg. The increased metabolic needs of muscle and its lower vascular resistance after exercise result in the decreased volume of blood available, preferentially perfusing the muscular bed at the expense of the skin where the peripheral resistance remains essentially unchanged. As muscle metabolism recovers and its arterial resistance increases, blood is redirected towards skin and $PtcO_2$ rises towards resting levels. On a physiological basis it would therefore appear that measurement of the changes in skin oxygen tension that reflect alterations in blood flow to the skin should be a sensitive method of diagnosing arterial insufficiency. It may also diagnose it at an early stage when resting perfusion is normal.

The question should be asked as to what advantage $PtcO_2$ has over conventional noninvasive vascular testing such as segmental ankle pressures at rest and following exercise. Segmental ankle pressures are of limited value, particularly where multi-level disease is present. The difficulty of placing the thigh cuff high enough on the thigh renders it difficult to separate iliac or common femoral artery occlusion from complete occlusion of the superficial femoral artery. In patients with calcified or rigid vessels, as seen in very old or diabetic patients, falsely high values ankle pressures may be obtained. Earlier experience in our laboratory has shown a correlation coefficient of 0.64 ($p < 0.01$) between $PtcO_2$ and ankle pressure readings with the $PtcO_2$ being more sensitive and specific.[10]

In practice, the measurement of $PtcO_2$ is a simple procedure with many advantages. It does not require extensive training to calibrate or use the equipment. Because the instrument provides a quantitative reading, there is little likelihood for observer error or variation. The mea-

TABLE 4. *Ranges and Means of $PtcO_2$ in Patients with Nonvasculogenic Leg Pain*

	Supine	Erect	Exercise	1 Min. Postexercise	5 Min. Postexercise	10 Min. Postexercise
Above knee	64.4 ± 7.27* (53–79)	72.2 ± 4.93 (63–82)	77.8 ± 6.97 (67–90)	73.5 ± 5.93 (60–84)	71.0 ± 4.86 (64–82)	67.4 ± 6.69 (58–83)
Below knee	60.9 ± 6.16 (50–72)	70.7 ± 5.42 (62–78)	76.3 ± 8.84 (64–90)	69.4 ± 7.74 (51–82)	67.4 ± 7.07 (50–77)	63.8 ± 9.25 (50–84)
Foot	57.7 ± 5.82 (51–68)	68.9 ± 6.39 (59–80)	74.0 ± 9.16 (59–88)	66.4 ± 6.10 (53–78)	66.3 ± 6.88 (52–79)	61.6 ± 7.94 (50–82)

Values expressed in mmHg.

* Standard Deviation.

surement is made noninvasively and patients do not experience discomfort from the electrodes.

The disadvantages of this technique are few. The electrodes may sometimes come loose, particularly during exercise. This can be quickly recognized by a sudden increase in $PtcO_2$ readings to over 100 mmHg. This problem can be prevented by ensuring that the electrode is adequately secured to the skin and by taking care to prevent the patient from pulling on the cables when exercising. Compared with conventional pre- and postexercise ankle pressures, each complete test takes about 15 minutes longer.

Conclusion

This study has demonstrated that $PtcO_2$ is a safe, simple, reproducible, noninvasive way of assessing the peripheral circulation. When performed in the manner described by us, $PtcO_2$ is capable of noninvasively distinguishing patients with arterial insufficiency from patients with a normal peripheral circulation with a high degree of sensitivity and specificity. The results of $PtcO_2$ correlate well with angiographic findings and with the expected physiological findings.

Summary

Transcutaneous oxygen measurements were made at three points on the leg in 174 people; 36 were controls and 138 were undergoing investigation for peripheral vascular disease. It was found that there was some overlap in the range between controls and patients with vascular disease, but the latter could be distinguished from patients without vascular disease by observing the $PtcO_2$ responses to exercise. There was a difference in the recordings obtained from the two groups in that, following exercise, the $PtcO_2$ in normal patients slowly declined towards resting values, while in patients with vascular disease it underwent a sharp decline below the level of the resting value. This decline produces a characteristic tracing that can be used to differentiate patients with vascular disease. In cases where angiogrpahy was performed there was complete agreement between the two tests, given $PtcO_2$ a sensitivity of 100% and a specificity of 100% in the diagnosis of peripheral arterial insufficiency.

TABLE 5. *Factors Affecting Tissue Oxygen Tension*

A. Systemic
1. Arterial oxygen content
a. Inspired oxygen concentrations
b. Ventilation
c. Lung Function
d. Hemoglobin level
e. Hemoglobin saturation
f. hemoglobin affinity for oxygen
2. Blood flow
a. Cardiac output
b. Perfusion distribution
B. Local
1. Skin thickness
2. Capillary formation and density
3. Oxygen consumption of skin
4. Inflammation, edema, etc.

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